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Expression and characterization of cM-T413, a chimeric anti-CD4 antibody with in vitro immunosuppressive activity.

Looney JE, Willinger A, Lin G, Rieber EP, Riethmuller G, Ghrayeb J.

Department of Molecular Biology, Centocor, Inc., Malvern, Pennsylvania 19355.

Anti-CD4 monoclonal antibodies (mAbs) have shown considerable promise in the treatment of rheumatoid arthritis, psoriasis, and allograft rejection and may have potential use in blocking HIV-1 infection. One such anti-CD4 mAb we have developed, chimeric M-T412 (or cM-T412), has been used in clinical trials to treat rheumatoid arthritis, generalized postular psoriasis, and other autoimmune diseases. Here we report the cloning and expression of a second chimeric anti-CD4 mAb using M-T413, a murine mAb that blocks HIV-1 infection of H9 cells. We cloned the immunoglobulin light and heavy chain variable regions of M-T413, combined them with the human kappa (light chain) or G1, G2, G3 and G4 (heavy chain) constant regions in human expression vectors, and expressed these chimeric mAbs in 653 cells. Like chimeric M-T412 IgG1, the chimeric M-T413 mAbs inhibit T-cell proliferation in the mixed lymphocyte response and thus can act to immunosuppress CD4+ T-cell response. In contrast to M-T412, however, the M-T413 chimeric mAbs have reduced activity in an antibody-dependent cell-mediated cytotoxicity (ADCC) assay using human CD4+ target and effector cells. We conclude that the chimeric M-T413 mAbs have potential utility in treating autoimmune disease and may be useful as prophylactics in preventing HIV-1 infection.

PMID: 8081558 [PubMed - indexed for MEDLINE]

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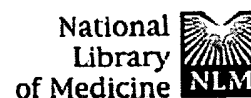
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Prevention of lethal graft-versus-host disease in mice by monoclonal antibodies directed against T cells or their subsets. I. Evidence for the induction of a state of tolerance based on suppression.

Knulst AC, Tibbe GJ, Noort WA, Bril-Bazuin C, Benner R, Savelkoul HF.

Department of Immunology, Erasmus University, Rotterdam, The Netherlands.

Lethal GVHD in the fully allogeneic BALB/c (donor)-(C57BL x CBA)F1 (recipient) mouse strain combination could be prevented by a single dose of IgG2b monoclonal antibodies (moAb) directed to T cells. The influence of the time of administration of this moAb after GVHD induction and the effect of anti-T cell subset moAb on the development of GVHD was investigated in this study. Moreover, the state of tolerance in the mice that had become long-term chimeras was examined. Anti-Thy-1 treatment of the recipients 1 day before, 2 h before or 1 day after reconstitution almost completely prevented lethal GVHD. A single dose of 100 micrograms of anti-Thy-1 was as effective as four daily doses of 25 micrograms each. Treatment with a single dose of 25 micrograms or with intervals of 4 days between doses of 25 micrograms was statistically significantly less effective. We injected the recipients with moAb directed to the CD4+ or CD8+ T cells subsets. Using a dose of 100 micrograms moAb, anti-CD4 treatment appeared to be less effective than anti-Thy-1 treatment whereas anti-CD8 treatment was not effective at all. A double dose of anti-CD4 was equally effective as anti-Thy-1 treatment. All mice that became long term survivors remained free of signs of GVHD and were > 99% repopulated with donor type cells. Injection of spleen cells from these BALB/c into (C57BL x CBA)F1 chimeric mice was used to reconstitute lethally irradiated BALB/c, BALB.K and (C57BL x CBA)F1 recipients. Lethal GVHD developed in the BALB.K and (C57BL x CBA)F1 recipients but not in the BALB/c recipients.(ABSTRACT TRUNCATED AT 250 WORDS)

PMID: 8199571 [PubMed - indexed for MEDLINE]